days later. However, she continued to bring up thick, tenacious mucus with some difficulty because of a weakened cough. On August 23, she became febrile and dyspneic and upon physical examination dullness to percussion and diminished breath sounds were noted in the right lower lung field, and a density of the lower two-thirds of the right side of the chest was noted on an x-ray film. Bronchoscopy was done the next day and a large amount of thick, tenacious mucus was removed from the right lower lobe bronchus. The patient felt improved after this procedure, but there was essentially no change by auscultation and percussion and by x-ray. Following antibiotic therapy she recovered completely.

SUMMARY

This report concerns a protracted non-fatal form of botulism with severe weakness of muscles and respiratory paralysis apparently due to the ingestion of an extremely minute quantity of the botulinus toxin. Tracheotomy and use of a respirator probably were life-saving. Paralysis of the pharyngeal and respiratory muscles closely allies this disease to bulbar poliomyelitis insofar as management and treatment of the patient is concerned. In spite of the complicating atelectasis and pneumonia, the patient recovered completely.

1200 North State Street.

Familial Periodic Paralysis

JOHN L. DENNEY, M.D., Los Angeles

FAMILIAL PERIODIC PARALYSIS was first reported by Musgrave[4] late in the 17th Century. The familial characteristics were first described in 1882. The observation that serum potassium content decreased during an attack was reported by Biemond and Daniels in 1934.

Characteristically the onset is noted in adolescence, although it has been reported as early as the sixth month of life and as late as the sixth decade. Attacks usually occur at night during sleep. They may be precipitated by a heavy meal, by violent exercise or by exposure to cold. Attacks vary in severity from patient to patient and from time to time in the same patient. The attacks may range from a mild weakness of a single muscle group to complete quadriplegia with respiratory paralysis and death. The cranial nerves are usually spared. The disease usually becomes less severe with age.

REPORT OF A CASE

A 15-year-old Caucasian girl was referred to the Communicable Disease Unit of the Los Angeles County General Hospital May 6, 1953, with a diagnosis of poliomyelitis and a history of paralysis of six hours' duration involving all four extremities.

The patient had awakened at 4 o'clock on the morning of entry, unable to move and with complaint of stiffness of the neck without headache. Upon admittance she was alert, afebrile and beginning to recover ability to move her arms. Upon physical examination, complete paralysis of arms and legs with pronounced paresis of the hands and feet was noted. The neck was supple but weak. Deep tendon reflexes were absent throughout. The spinal fluid was clear; there were no cells. Reaction to a Pandy test showed a trace of protein; the sugar content was normal. A serum potassium determination on blood taken at the time of admission revealed a level of 2.1 mEq. per liter. An electrocardiogram was consistent with hypopotassemia.

The patient recalled three to four similar previous episodes during the preceding two years, all occurring at night. From members of the family it was learned that the patient's great-great-grandfather, great-grandfather, grandfather, father, four of six uncles (father's brothers), none of four aunts, and two of the patient's siblings had had similar episodes.

The patient's father had had the most severe disease of the group; attacks occurred regularly after severe exercise and were frequently precipitated by a heavy meal.

The patient gradually regained function during the 24 hours after admittance and the remainder of the stay in hospital was uneventful. An attempt to precipitate an attack with a glucose tolerance test was unsuccessful. Serum potassium remained within normal limits during the test. An electroencephalogram was normal.

SUMMARY

A case of familial periodic paralysis is reported. Although previously undiagnosed, the disease was traced through five generations of the patient's family.

1200 North State Street.

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Communicable Disease Unit, Los Angeles County General Hospital, service of A. G. Bower, M.D.